Metabolic Acidosis in Restraint-associated Cardiac Arrest: A Case Series

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Abstract. The mechanism of death in patients struggling against restraints remains a topic of debate. This article presents a series of five patients with restraint-associated cardiac arrest and profound metabolic acidosis. The lowest recorded pH was 6.25; this patient and three others died despite aggressive resuscitation. The survivor's pH was 6.46; this patient subsequently made a good recovery. Struggling against restraints may produce a lactic acidosis. Stimulant drugs such as cocaine may promote further metabolic acidosis and impair normal behavioral regulatory responses. Restrictive positioning of combative patients may impede appropriate respiratory compensation for this acidemia. Public safety personnel and emergency providers must be aware of the life threat to combative patients and be careful with restraint techniques. Further investigation of sedative agents and buffering therapy for this select patient group is suggested. **Key words:** metabolic acidosis; restraint—physical; heart arrest; cardiac arrest. ACADEMIC EMERGENCY MEDICINE 1999; 6:239–243

MANAGEMENT of violent and agitated patients is a significant problem for public safety and emergency medical agencies, and death occasionally occurs during restraint of these patients. The cause of death in these circumstances has not been elucidated. Speculation has focused on autonomic reflexes, restraint stress,[1] agitated delirium from stimulant drugs,[1] and positional asphyxia due to use of the hobble (or "hog-tie") position as potential contributing factors.[2–4] Acidosis has not been cited as a contributing factor in restraint-associated death. Severe metabolic acidosis has, however, been noted with stimulant drug use (notably cocaine),[5] and when exertion and stimulant drug use are combined.[6–8] Profound metabolic acidosis can have significant negative cardiovascular effects, including promotion of dysrhythmias and autonomic instability, which may contribute to cardiovascular collapse.

We present five cases of cardiovascular collapse occurring in ED patients who were struggling despite maximal restraint techniques. They were all profoundly acidotic. Our experience with these cases led to institution of an aggressive proactive approach in the medical management of these patients, and to changes in local police restraint practices. An additional five patients are reported who were treated with this proactive approach.

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Received August 4, 1998; revision received October 20, 1998; accepted November 2, 1998.
Supported by the Department of Emergency Medicine Research Fund.
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CASE 1

A 36-year-old man was acting extremely agitated and belligerent on a downtown sidewalk. When approached, he attacked a police officer and ran. He was subsequently subdued by several officers. He was transported to the ED, where he continued to fight vigorously while lying prone with his hands cuffed behind him. Breath analysis was negative for ethanol. Shortly thereafter, the patient had a witnessed respiratory arrest. He was intubated within 4 minutes of his apnea. Shortly after intubation, a 15-second episode of asystole was noted; he recovered a sinus rhythm at a rate of 140 beats/min after epinephrine, atropine, and hyperventilation. His initial arterial blood gas (ABG) obtained 5 minutes after intubation was pH 6.46, pCO_2 49 mm Hg, pO_2 523 mm Hg, and a bicarbonate (HCO_3) of 4 mEq/L. Aggressive fluid resuscitation was begun with crystalloid and 100 mEq of sodium bicarbonate was given. Within 30 minutes, the patient awoke and was able to follow commands, but had a bilateral lower-extremity paraplegia. The patient was admitted to the intensive care unit (ICU). His serum lactate level obtained one hour after admission was more than 24 mEq/L. He was extubated the following day, by which time his paraplegia had completely resolved. His urine toxicology screen was positive for cocaine and he admitted to being cocaine-intoxicated during his arrest. He ruled out for a myocardial in-

farction by creatine kinase (CK) isoenzymes, but had a several-day course of rhabdomyolysis with peak CK levels higher than 40,000 IU/L. He was aggressively hydrated and maintained a good urine output. He developed renal insufficiency with a peak creatinine of 3.7 mg/dL. He also developed evidence of liver injury with elevated liver transaminases and increased prothrombin and partial thromboplastin times. These abnormalities resolved after several days. He was discharged on hospital day 5 with good urine output and a creatinine of 3.2 mg/dL. He failed to keep his follow-up appointments.

CASE 2

A 39-year-old man with a history of unspecified psychiatric illness was brought to the emergency psychiatric area for evaluation of agitation and psychosis. The patient became violent and was restrained by several security guards. He was placed prone with his arms behind him. During the restraint process he became apneic and pulseless. He was moved across the hallway to the stabilization room in the ED and immediately intubated. He was initially in a nonperfusing bradycardia, which deteriorated into ventricular fibrillation, and then asystole. An external pacemaker was applied but failed to capture. Epinephrine, atropine, and bicarbonate were given without results. A thoracotomy was then performed, and after internal cardiac massage and defibrillation, he developed a perfusing rhythm. The ABG values obtained immediately after thoracotomy were pH 6.81, pCO_2 30 mm Hg, and pO_2 162 mm Hg. His initial anion gap was 37 mEq/L. He was given a total of 450 mEq of bicarbonate during the case. His hemodynamic status stabilized and the acidosis reversed within 12 hours. Life support was subsequently withdrawn two days later due to a persistent vegetative state. Urine toxicology was positive for cocaine; serum levels were not available.

CASE 3

A 30-year-old man stole a purse, and after a long foot chase was apprehended by two witnesses who sat on the patient to restrain him. He lost consciousness, and when the paramedics arrived, he was in cardiac arrest with an idioventricular rhythm. He was intubated orally in the field, CPR was begun, and over the next 10 minutes, he was given a total of 4 mg of epinephrine, 1 mg of atropine, and 0.4 mg of naloxone. No bicarbonate was given. He was transported to the ED, where the idioventricular rhythm became asystolic. Initial ABG levels obtained at ED arrival showed pH less than 6.8, pCO_2 18 mm Hg, and pO_2 255 mm Hg. Serum bicarbonate was undetectable by standard assays. He received 150 mEq of sodium bicarbonate, escalating doses of epinephrine to 5 mg, and an additional 1 mg of atropine. An external pacer failed to capture. Transthoracic echocardiography showed a motionless heart, and resuscitative efforts were halted. Urine toxicology was positive for cocaine; serum levels were unavailable.

CASE 4

After firing a gun in an apartment, a 39-year-old man was apprehended and restrained by several police officers. He continued to struggle during transport in a prone position with his hands cuffed behind his back. Upon entering the ED, he violently kicked a door, and then had a sudden cardiopulmonary arrest. CPR was started, and the patient was moved to the stabilization area. The presenting rhythm was idioventricular. Despite epinephrine, atropine, and standard advanced life support, he failed to respond, and died. Initial ABG levels immediately upon ED arrival were pH less than 6.8, pCO_2 more than 100 mm Hg, and pO_2 30 mm Hg. The anion gap was 24 mEq/L. Serum bicarbonate was undetectable by standard assays. A serum toxicology screen was positive for free cocaine. Autopsy revealed a nonthrombosed 75% left anterior descending coronary artery stenosis. Cause of death was attributed to "cocaine-induced excited delirium."

CASE 5

A 38-year-old man was observed standing in the middle of a local street, attempting to hit passing cars with his fists. Responding police personnel were not able to escort him from the street. The patient was wrestled to the ground, maced, and then carried to the median and placed on his side. He continued to struggle, then had a sudden cardiorespiratory arrest. Paramedics were present and immediately intubated the patient. The initial rhythm was ventricular fibrillation, with defibrillation resulting in asystole. The patient received 1 mg of epinephrine, 1 mg of atropine, and 50 mEq of sodium bicarbonate with a transient return of pulses at a rate of 120 beats/min. He became pulseless again. Five milligrams of epinephrine and an additional 50 mEq of sodium bicarbonate were given, again with return of pulses. The patient was transported to the ED where initial ABG levels were pH 6.25, pCO_2 50 mm Hg, pO_2 221 mm Hg, and bicarbonate 4 mEq/L. He was admitted to the ICU, where he required aggressive fluid and pressor support. Refractory hypotension and disseminated intravascular coagulation led to an eventual

bradyasystolic arrest ten hours after admission. Resuscitative efforts were futile. Autopsy showed no anatomic cause of death. Serum toxicology revealed rising levels of benzoylecgonine, a cocaine metabolite, during the time from ED presentation until the patient's death.

DISCUSSION

The phenomenon of sudden death in restraints has long been recognized. The physiologic derangements that lead to death are still poorly understood. In 1985, Wetli and Fishbain described seven cocaine-intoxicated patients who were apprehended because of violent or bizarre behavior, who then went on to have sudden death.[1] While they stated that the cause of death in these cases of "excited delirium" is unknown, they speculated on possible causes such as autonomic reflexes, arrhythmias, or restraint stress. Much like our cases, each of the seven patients experienced extreme exertion while either fleeing or fighting vigorously while being subdued.

In 1990, Bethke et al.[7] described a case of cocaine ingestion and significant exertion in a patient who was chased and shot at with a shotgun. He suffered only minor superficial pellet injuries, and had minimal blood loss. His initial pH was 6.91, pCO_2 30 mm Hg, pO_2 155 mm Hg, and bicarbonate 6 mEq/L. His lactic acid level was 29 mEq/L, with an anion gap of 38 mEq/L. He was never restrained, but exertion and cocaine use contributed to a substantial metabolic acidosis. Buzzuto[8] also reported a case of lactic acidosis secondary to cocaine use and exertion. This patient presented with an initial pH of 6.96, pCO_2 25 mm Hg, pO_2 125 mm Hg, bicarbonate 6 mEq/L, and anion gap 34 mEq/L. He received 2 liters of normal saline and 100 mEq of sodium bicarbonate. His lactate was 4.2 mEq/L one hour after the above therapy. Both patients made full recoveries.

Our case series describes five patients who were combative despite maximal restraint techniques and had subsequent cardiorespiratory arrest with profound metabolic acidosis. Three, and possibly more, of our patients had used cocaine proximate to the inciting event. Unfortunately, serum toxicology screens were not available for two of our cases. Lactate levels were obtained in only one case; no other cause of metabolic acidosis was evident in these cases. No case of restraint death has been seen at our institution in which the patient was not acidotic. No significant electrolyte abnormality was present, the highest potassium level in the group was 6.0 mEq/L, but most were between 3.5 and 4.5 mEq/L.

Simple exertional lactic acidosis does not usually reach the levels attained by the patients described in our series. Bruce et al. studied lactic acid production in athletes and found levels of 7.5 to 10.7 mEq/L after strenuous exercise.[9] In an article reviewing lactic acid production in athletes, Giammarco noted a peak lactate level of 17 mM/L in sprint-trained, and 12.6 mM/L in endurance-trained athletes.[10] These levels are much lower than the very high levels noted in case 1 (more than 24 mEq/L) and in Bethke et al.'s case report.[7]

The degree of acidosis in our patients also exceeds that routinely seen in sudden death. In Dybvik et al.'s series of 257 patients with out-of-hospital arrests receiving no buffer therapy, the average pH at hospital arrival was 7.23, with a range of 7.20–7.26.[11] The highest pH in our series was 6.81.

Several factors may contribute to the profound acidosis we observed in our cases. There may be exacerbation of exercise-induced lactic acidosis by sympathetic-induced vasoconstriction, enhanced by the actions of cocaine in at least some cases.[8] Bethke et al. and Buzzuto theorized that such vasoconstriction might impede clearance of lactate by the liver, thus extending the time to recovery.[7,8]

Second, psychosis and delirium, including that due to drug use, may alter pain sensation, thus allowing exertion far beyond normal physiologic limits. This might result in a severe acidosis with maximal sympathetic discharge. Subsequently, sympathetic output may be suddenly reduced due to catecholamine depletion or the onset of obtundation and body relaxation. Either could result in a loss of vascular tone, with peripheral muscle lactate mobilization and sudden cardiovascular collapse. Acidosis of this magnitude should stimulate a compensatory increase in respiratory drive; however, reflex compensation may be limited by positions that reduce respiratory efficiency.

The method of restraint called "hog-tying," or hobbling (in which ankles and wrists are bound together behind the back), or even prone placement may impede a patient's ability to develop a compensatory respiratory alkalosis. Reay et al.[3] and Bell et al.[4] have described the phenomenon of "positional asphyxiation" in which patients suffer a cardiac arrest while in a hobbled position. Stratton et al. described two stimulant-intoxicated patients who suffered respiratory arrest while in hobble restraints.[2] However, research by Chan et al. has cast doubt on the asphyxiating effects of the position itself. In a crossover study of 15 volunteers who had exercised vigorously for 4 minutes, the maximal ventilatory volume was 20% lower in the restrained vs sitting unrestrained position, but there was no difference between the positions with respect to pO_2, pCO_2, or oxygen saturation.[12] Though restraints may not have significant impact

TABLE 1. Patients Treated Who Did Not Sustain Cardiopulmonary Arrest

	ABGs*	Situation	Treatment	Complications
Case 1	6.76/158/22/3	Jumped out of a first-story window, ran four blocks, extremely combative. Heroin and crack used. Lactate >24 mEq/L.	Five ampules $NaHCO_3$, IV fluids, lorazepam, paralysis, intubation	Presented in atrial fibrillation, resolved; discharged 48 hours later
Case 2	7.12/143/22/7	Yelling in street, fought with officers. Admits crack cocaine use.	Droperidol 10 mg IV, IV fluids	None; discharged 12 hours later
Case 3	7.02/130/23/6	Out of control in store, fought with officers, kicked out police car window. Admits crack cocaine use.	Droperidol, ketamine, $NaHCO_3$, intubation	Creatine kinase peak 5,016 IU/L; mild congestive heart failure—resolved; left against medical advice 24 hours later
Case 4	7.16/92/27/9	Jumped out of a second-story window, ran into street, hit by car, restrained by neighbors. Calming on ED arrival. Admits crack cocaine use.	Aggressive IV fluids	Discharged next day
Case 5	7.00/154/31/8	Struck with hammer to head in fight, calming on arrival. Admits crack cocaine use.	IV fluids, $NaHCO_3$ 2 ampules	Hypotension—systolic blood pressure 70 mm Hg—reversed with fluids; discharged next day

*ABGs = arterial blood gases: pH/pO_2 (in mm Hg)/pCO_2 (in mm Hg)/HCO_3 (in mEq/L).

on healthy people even post-exercise, reduction in ventilatory volume by 20% may significantly impact the ability of an acidotic patient to develop a compensatory respiratory alkalosis. The restraint process itself may increase mortality, possibly because efforts at physical restraint often provoke still further struggle, increasing acid production in already compromised patients.

Finally, the combination of cocaine and restraint seems to have a deleterious effect on patient outcome. In a study by Pudiak and Bozarth, rats were given cocaine daily, then placed for 30 minutes in a restraint cylinder that allowed movement, but not freedom to turn around. The experimental group had a 50% mortality by day 3 (half of these deaths occurring on the first day), compared with 8% in a cocaine/no restraint control group, and no mortality in a restrained saline placebo group.[13] Chronic cocaine use may further predispose individuals to develop fatal excited delirium, though the mechanism remains to be elucidated.[14]

Continued combativeness despite restraints, especially in the setting of sympathomimetic agents such as cocaine, seems to be a marker for a patient at high risk for death, regardless of pathophysiology. Physical restraint techniques may provoke further struggle, with possible development of overwhelming acidosis. Out-of-hospital use of chemical sedatives should be studied in selected patients to potentially prevent this chain of events.

Locally, police agencies have been trained to restrain patients in a side-lying position when possible, and to avoid cuffing a patient's hands behind the back in a prone position. Also, continued struggling against restraints is recognized as a medical emergency, and emergency medical services (EMS) personnel respond in these situations. Once in the ED, aggressive sedation and correction of pH with sodium bicarbonate and hyperventilation are practiced at our institution.

As our awareness of these cases has grown, we have treated at least five additional patients who continued to struggle against restraints until ED presentation (Table 1). All admitted crack cocaine use once their mental status normalized. None of these patients experienced cardiac arrest, although one was hypotensive, with a systolic blood pressure of 70 mm Hg at presentation. Initial pH ranged from 6.76 to 7.16. These patients were treated with aggressive fluid resuscitation and those with pH < 7.10 received sodium bicarbonate. Aggressive sedation with droperidol, benzodiazepines, and, in one case, ketamine was practiced. All of these patients resolved their acidosis and four were discharged or left within 24 hours. One patient was kept for 48 hours for cardiac monitoring. Despite these out-of-hospital and hospital efforts, deaths continue to occur, as evidenced by case 5 in our original series (above).

CONCLUSION

These cases suggest that a profound metabolic acidosis is associated with cardiovascular collapse following exertion in a restrained position. It is important that public safety personnel recognize the significant life threat when a patient remains combative despite restraints. Avoiding the hobble restraint position and emphasizing side rather than prone positioning may eliminate some of the problems that contribute to the pathophysiology of such

deaths. Early involvement of EMS may help to prevent in-custody deaths, particularly if chemical restraint or bicarbonate therapy can be used. Good outcomes are still possible despite the pronounced level of acidemia; aggressive hyperventilation and bicarbonate therapy may offer the best chance of patient survival. More study is needed both on the physiologic changes of restraint positioning and on the mechanisms of death in these cases in order to better manage this challenging group of patients.

The authors gratefully acknowledge Katherine Berg, MD, Hennepin County Medical Examiner's Office, and Charles Wetli, MD, Suffolk County (FL) Medical Examiner's Office, for their assistance and suggestions.

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